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Respiratory failure associated with hypoventilation in a patient with severe hypothyroidism

Munehisa Fukusumi, Toshiko Iidaka, Atsuto Mouri, Yoichiro Hamamoto & Mitsuhiro Kamimura

Department of Pulmonary Medicine, National Hospital Organization Disaster Medical Center, Tokyo, Japan

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Correspondence
Munehisa Fukusumi, Department of Pulmonary Medicine, National Hospital Organization Disaster Medical Center, 3256 Midori-cho Tachikawa, Tokyo, Japan. E-mail: munesumi@hotmail.com

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Abstract

A 70-year-old Japanese man was admitted to hospital because of decreased consciousness due to type II respiratory failure. Severe hypothyroidism was diagnosed and considered to be associated with hypoventilation due to respiratory muscle dysfunction and sleep apnea syndrome. His status was improved partially by replacement of thyroid hormone. Despite maintaining a euthyroid state, improvement of respiratory muscle dysfunction was incomplete.

Introduction

The mechanisms of type II respiratory failure due to hypothyroidism include (1) impaired central ventilator responses to hypoxia and hypercapnia; (2) hypoventilation caused by respiratory muscle dysfunction; and (3) obstructive sleep apnea syndrome (OSAS) [1]. Here, we report an elderly Japanese man who suffered from severe ventilatory failure associated with hypothyroidism.

Case Report

A 70-year-old man with a past history of hypertension presented with decreased consciousness. The patient had begun to have easy fatigability one year prior to admission. Speech and actions became slower accompanied by excessive sleepiness in daytime and snoring during sleep. He had smoked one pack daily for 40 years. On the morning of admission, he became unresponsive and was brought to the emergency room. On examination, his body mass index was 16.5, Glasgow Coma Scale score was 6, blood pressure was 178/78 mmHg, heart rate was 89/min, respiratory rate was 16/min, and temperature was 37.9°C. Head and neck examination demonstrated no goiter or thyroid bruit. Cardiac, respiratory, and abdominal examination was normal. His hemoglobin was 11.9 g/dl, creatine kinase was 532 IU/L, and plasma glucose was 115 mg/dl. Arterial blood gas analysis in room air showed severe hypercapnea (partial pressure of CO_2 157.5 mmHg) with a compensatory increase of bicarbonate (HCO_3^-). Chest computed tomography showed no emphysematous changes, but there were bilateral pleural effusions and passive atelectasis with an air bronchogram in the left lower lobe. Echocardiography revealed no abnormality. Mechanical ventilation was introduced. Five days after admission, he was diagnosed as having primary hypothyroidism (tyroid stimulating hormone [TSH] 111.3 μIU/mL, free thyroxine [fT] 3 0.0 pg/mL, fT 4 0.2 ng/dL). Thus, we diagnosed respiratory failure associated with hypothyroidism. Oral levothyroxine was started under steroid cover and the patient became euthyroid rapidly. Ten days after admission, the patient was extubated onto noninvasive positive pressure ventilation (NIPPV). Subsequently, severe sleep apnea syndrome with an apnea hypopnea index of 45.8 was found by polysomnography. Fifty days after admission, he was discharged, but dependent on NIPPV during the night (Fig. 1). Respiratory function tests after admission showed a restrictive ventilatory impairment with vital capacity of 1.52 L (47.9% predicted) and forced expiratory volume of 0.52 L in 1 sec. Diffusing capacity for carbon monoxide

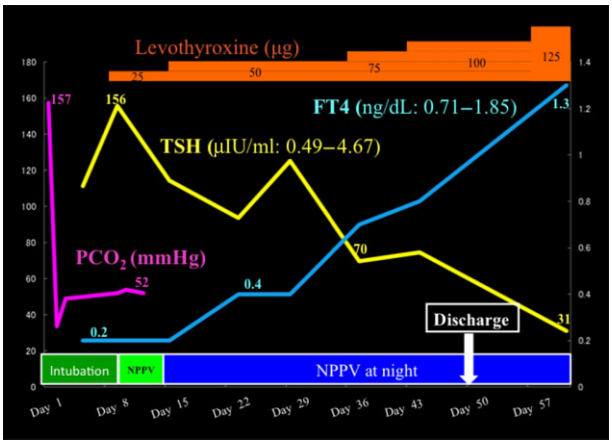

Figure 1. A clinical course in the acute phase. FT4, free thyroxine 4; NNPV, noninvasive positive pressure ventilation; PCO_2, partial pressure of CO_2; TSH, thyroid-stimulatinghormone.

to alveolar volume was 5.52 mL/min/mmHg/L (129% predicted). Electromyography showed no myogenic change and there was also no abnormality on brain magnetic resonance imaging examination. Despite maintaining a euthyroid state, his restrictive ventilatory defect progressed gradually. He became almost continuously dependent on NIPPV 6 years after.

Discussion

Although rare, hypothyroidism can cause respiratory disorders via three mechanisms. The first feature is an impaired central ventilatory response to hypoxia and hypercapnia. Some investigators have reported that approximately half of the hypothyroid patients have an attenuated increase in minute ventilation in response to rising CO_2 and nearly all have a similar impairment to reduced O_2 tensions [2]. A second feature is hypoventilation caused by weakness of diaphragm and other respiratory muscles, which occurs in 30%–40% of hypothyroidism. Khaleeli and Edwards reported that hypothyroidism causes demyelination and fibrosis of the phrenic nerve, irreversible type II fiber atrophy, and loss of total skeletal muscle mass [3]. The prognosis of respiratory muscle dysfunction depends on the degree of hypothyroidism, not its duration. Ladenson et al. found a TSH higher than 90 μIU/mL to be a poor prognostic factor [4], as found in our case. A third feature is OSAS, occurring in 25%–35% of subjects suffering from hypothyroidism. The main pathophysiology of OSAS is likely to be pharyngeal narrowing due to soft tissue infiltration by mucopolysaccharides and protein [5]. The swelling-related impairment of muscle contraction and relaxation may affect the activity of the upper airway dilatator muscles during night. Central sleep apnea may occur due to blunted ventilatory and neuromuscular response to hypoxia, although obstructive apnea usually predominates.

After treatment, hypercapnic and hypoxic respiratory responses usually normalize after 7–14 days of hormone replacement [1]. However, respiratory muscle dysfunction may either be permanent or take months to improve [3]. Ventilation-assist therapy, including NIPPV, may be necessary in acute severe hypothyroidism [1] and may improve the quality of life in chronic phase as a palliative therapy. Nutrition therapy and physiotherapy are also necessary to prevent further muscular atrophy.

In conclusion, hypothyroidism can cause the respiratory failure associated with hypoventilation and sleep apnea syndrome. Although it is rare, hypothyroidism should be considered as a cause of type II respiratory failure.

Disclosure Statements

No conflict of interest declared.

Appropriate written informed consent was obtained for publication of this case report and accompanying images.

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